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Editorial

It's not all about the knee adduction moment: the role of the knee flexion moment in medial knee joint loading

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It is a longstanding tenet that mechanical loading plays a central role in the pathogenesis of osteoarthritis (OA), yet the precise loading mechanism responsible is not clearly understood[1]. The contact force acting on articular cartilage has been flagged as a likely candidate, and given the high prevalence of medial tibiofemoral OA, attempts have been focussed on characterising articular cartilage contact force in the medial tibiofemoral compartment (MCF). Two different approaches have been adopted in this endeavour: (1) direct measurement of MCF in individuals with instrumented knee joint replacements, and, (2) biomechanical modelling to estimate MCF *in vivo*, i.e., in a native joint. Data from such studies can provide us with an indication of the role of MCF in disease pathogenesis, and how MCF is influenced by various interventions. Unfortunately however, these approaches are either (1) invasive in nature, or (2) computationally expensive and/or complex, and thus of limited feasibility for widespread use.

Enter clinical gait analysis: a non-invasive, relatively low cost, system consisting of cameras and force plates (that measure the forces imparted upon the body from the ground), that can calculate the net external forces acting on the joints of the body, such as the knee. As such, these systems are widely available in both the research and clinical environment. Clinical gait analysis techniques, for example, were used in the seminal work by Miyazaki and colleagues[2], to identify the frontal plane turning force at the knee (the knee adduction moment) during walking as the strongest mechanical risk factor for medial knee OA disease progression, i.e., over and above the effect of bodyweight and alignment.

The turning forces, or moments, acting at the knee have an intuitive relationship with the MCF. The knee adduction moment (KAM) represents the moment acting on the joint in the frontal plane, and during walking it typically acts to rotate the tibia medially on the femur. Thus, it would be expected – and has been shown – that with a greater KAM the MCF increases[3,4]. Further, it has been argued that the external moment acting on the knee in the sagittal plane – the knee flexion moment (KFM) – may influence MCF, and subsequently the risk of OA incidence and progression. To balance the external KFM that occurs during gait the quadriceps contract to produce an internal knee extension moment. When activated, the quadriceps also impart a compressive force across the tibiofemoral joint; hence the postulated relationship with MCF. That being said, the relative contribution of the knee moment in the frontal (KAM) and sagittal (KFM) planes to the MCF is not well documented.

With this in mind, Manal *et al.*[5] evaluated the ability of the KAM and KFM to predict peak MCF during straight line walking. A cohort of 10 patients, post ACL reconstruction – a population known to be at elevated risk of OA development – were evaluated. A biomechanical model was used to estimate muscle forces from electromyographic data, which, in turn, were used to estimate the MCF. Peak KAM and KFM were determined via an inverse-dynamics approach, as commonly used in clinical gait analysis (e.g.,[2,6]). Regression modelling was used to predict the variance in the peak MCF that was explained by the peak KAM, and secondly, the variance in the peak MCF explained by the peak KFM over and above that explained by the peak KAM.

The peak KAM, peak KFM and peak MCF all occurred at a similar time point during the stance phase of gait (i.e., 23–25%). As hypothesised, peak KAM alone was predictive of 63% of the variance in the MCF. This prediction was improved by the inclusion of peak KFM in the model, predicting 85% of the variance in peak MCF i.e., the KFM improved the prediction by 22%. An increase in the KAM or KFM was associated with an increase in the MCF. Interestingly, data from individual participants indicated that the contribution of the KAM and KFM to MCF varies substantially; that is, a participant with a small peak KAM may have a similar peak MCF to a participant with a large peak KAM. In the participant with the smaller peak KAM this may be explained by a higher peak KFM.

The finding that a higher KFM is predictive of a higher MCF was hypothesised, and, as such, not unexpected. Indeed, recent work in patients with instrumented knee replacements has demonstrated that the MCF is related to both the KAM and the KFM[7,8]. This is the first study however, to demonstrate the shared importance of the KAM and KFM in native knees, as opposed to in an artificial joint. Taken together, the consistent finding of the shared importance of the KAM and KFM in determining the MCF gives us some confidence that both of these indices should be considered when attempting to evaluate the magnitude of, or changes in, the MCF.

The interpretation of knee joint moments in estimating the MCF is as follows: a lower KAM (or a decrease in the KAM), does not necessarily equate to lower MCF (or a decrease in MCF). Assuming a high MCF is deleterious to joint health, this has implications for how interventions to modify joint load are interpreted. For example, Fregly and colleagues[9] investigated alternative walking patterns as a way to reduce the KAM, and demonstrated that with increased hip, knee and ankle flexion combined with altered

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pelvis kinematics, a decrease in the KAM was observed. This would suggest positive effects in terms of reduced MCF at first glance; however, they also report a concomitant increase in the KFM with a decreased KAM. Thus, the observed reduction in joint load, i.e., the MCF, may not be as substantial as originally assumed. Similarly, the peak KAM data from Hall and colleagues[6] effectively indicate no change in MCF at 2 years post meniscectomy. However, with a significant increase in the peak KFM over the 2 years it is likely there was an increase in MCF in these patients. Essentially, the take home message is that by using only the KAM as an index of knee load we may be misinterpreting effects on MCF. Considering the KAM in combination with the KFM is likely to provide us with a more accurate representation of any changes in loading on the medial compartment.

That the KFM, as well as the KAM, are predictive of MCF offers insight into interventions that are likely to be most effective in reducing the magnitude of the MCF. For example, external devices designed to redistribute load through the upper body (e.g., walking canes and poles) tend to be associated with decreases in both the KAM and KFM[10], and therefore may represent an effective approach to reduce MCF. Contrary to this, gait modifications employed to reduce the KAM tend to be accompanied by an increased KFM as they involve increased knee flexion[9,10]. Therefore, they may not be as effective in reducing joint load as they are in reducing the KAM. It is conceivable that alternative modifications to gait that reduce the KFM as well as the KAM are achievable. For example, a more “stiff knee” gait would tend to reduce the KFM[11], and combined with a moderate degree of trunk lean would likely also decrease the KAM. This is worthy of exploration.

In all of this, we should always be mindful of what the biomechanical measure is telling us with respect to patient health and function. The assumption here is that the MCF plays a causative role in medial compartment disease incidence and/or progression. While this makes sense from an intuitive perspective, there is no direct evidence to support this assumption. Some studies have shown the KAM to be a risk factor for disease progression; however, a meta-analysis of these data indicates no overall effect[12]. Importantly, a recent study published in *Osteoarthritis & Cartilage*[13] evaluated, for the first time, both the KAM (peak and impulse) and the KFM (peak) as risk factors for structural OA progression over 2 years. Contrary to expectations on the basis of the literature discussed thus far, the peak KFM was not shown to be predictive of disease progression. That said, if it is the MCF that is the underlying pathomechanical pathway driving the disease, attempts to reduce MCF via reductions in the KFM may still be beneficial. With advances in technology and refinement of biomechanical models, it would be reasonable to expect that prospective evaluation of MCF as a risk factor for OA disease progression is not too far away. This may represent a watershed in our understanding of the *in vivo* pathomechanics of knee OA.

Our knowledge of the relationship between external knee joint moments and MCF is currently based upon data from both instrumented knee replacements[7,8] and the native knees of ACL reconstructed patients[5]. We cannot be certain that the same relationship holds in other patient groups. With a similar magnitude of KFM, KAM and MCF in those with established medial tibiofemoral OA it is plausible that a similar relationship exists. Another important consideration is that our current knowledge is limited to the relationship between peak knee moments and peak MCF. Given that some evidence points towards the importance of total loading throughout stance upon disease progression (i.e., the impulse)[13,14], MCF impulse may play a pathomechanical role in OA. As the magnitude and direction of the flexion/extension moment at the knee varies substantially throughout stance, it is conceivable that the ability of the KFM to predict MCF impulse is markedly different from its ability to predict the peak MCF.

Knowledge that both the peak KAM and peak KFM are predictive of peak MCF has the potential to inform the development of interventions to reduce MCF, and, in turn, arrest the OA disease process. New evidence indicates that the peak KFM may not be an independent risk factor for OA disease progression. Should the KFM affect the underlying driver of the disease process (which may be MCF), attempts to reduce the KFM may slow the disease process. Of course, intervention to reduce the KFM, KAM and MCF will require evaluation through randomised controlled trials to ensure their biomechanical benefit translates to improved patient outcomes.

Conflict of interest statement

The author declares that they do not have any conflict of interest relating to this editorial.

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M.W. Creaby*
School of Exercise Science, Australian Catholic University, Queensland 4014, Australia

* Tel: 61 (0)7 3623 7587; Fax: 61 (0)7 3623 7650.
E-mail address: mark.creaby@acu.edu.au.

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